WAR MEDICINE AND SURGERY

Compiled under the direction of the Committee on the Survey of War Medicine of the National Health and Medical Research Council.

BLAST INJURIES AND CRUSH INJURIES.

BLAST INJURIES.

DURING, and subsequent to, the 1914-1918 war there were stories told of men killed in battle without showing an obvious wound, and some experiments on the effect of blast on animals were carried out to try to explain these observations. Until the time of the Spanish Civil War, however, there was no general realization of the fact that a man can be killed by the blast of a bomb without being injured externally. Once deaths of this kind had been reported, widespread interest was created, and, when large-scale bombing of England began, several clinicians feared that patients with blast injuries would be an important problem. This has not proved to be true: there have been relatively few patients suffering from the direct effects of blast, though many people have been killed by it indirectly. Whatever the final mortality and morbidity from blast prove to be, however, in time of war we cannot afford to neglect the study of the subject. Some of the research carried out has been done confidentially for the armed forces, but much of the fundamental knowledge about blast is available in medical literature.

The subject may be discussed from the following aspects: (a) physics of blast; (b) effects of blast on animals; (c) blast injuries in man; and (d) special features of immersion blast.

Physics of Blast.

When a bomb bursts, it produces three effects, which are included under the term "blast":

1. A wave of increased pressure moves outwards in all directions. The intensity of the pressure decreases rapidly at first as the wave progresses. The importance of this is that if a series of men were situated at slightly different distances from the site of explosion, there would be only a short distance between the last man to be killed and the first man to be uninjured; in other words, non-fatal injuries from blast are rather uncommon. These results are true, at any rate, of the smaller bombs, after the explosion of which the pressure falls rapidly to a harmless level. A similar reasoning may not be applicable to enormous modern bombs.

2. Following the wave of increased pressure there is a wave of decreased pressure, which is created as the compressed air recoils. It is obvious that the pressure can never be decreased more than fifteen pounds to the square inch below the normal value. The magnitude of this negative or suction wave is, therefore, not comparable to that of the positive wave, which rises to as much as one and a third tons to the square inch in the case of large modern bombs. The duration of the suction wave, however, is three to five times that of the pressure component, and it is well known that objects may resist the positive pressure but not the negative.

3. When an explosion occurs, the gases formed have to be dissipated, and they tend to move outward in the form of a wind. The wind follows immediately behind, but is distinct from, the positive pressure wave. The latter is a hydrostatic pressure, whereas the wind exerts its pressure in virtue of movement. It is the wind which is the shattering component of blast, and is responsible, for example, for blowing an object to pieces.

Blast waves are reflected like any other wave, and this causes one of their freak effects.[1][3] If an explosion occurs in a street lined with tall buildings, a series of waves is set up by reflection backwards and forwards between the two walls. The direct and the reflected waves may tend either to reinforce or to neutralize each other. Thus damage may be done throughout the street in what seems to be a bizarre manner. Some people who are relatively near the bomb may be protected by the interference effect and escape uninjured.

Another factor causing an apparent selection of victims is the irregular front of the blast wave. Because of this irregularity, people at equal distances from the bomb may be subjected to different pressures. Zuckerman quotes the following as a probable illustration of this: "In one case that was investigated eight men were crowded in a single story room, 20 feet by 16 feet, in which a bomb exploded slightly above floor level. Of the eight, five were killed and three survived."[5]

Again, many bombs penetrate the ground before exploding, so that a crater is formed and the blast is directed mainly upward. Anyone standing or lying near the lip of the crater may escape injury.

Effects of Blast on Animals.

The effect on animals varies, of course, according to their distance from the explosion. Those close to a powerful explosion are blown to pieces.

Further away there is a range of pressure, which varies from species to species, in which all animals will be immediately killed, without external injury, but often showing blood-stained froth or blood in the nose, mouth and upper respiratory passages. Still further away, and with considerable variation for different individuals, is a zone of pressure in which animals are found alive immediately after an explosion, but die at intervals, varying between a minute and a day. Animals in this group may have blood-stained froth in the upper respiratory passages and often suffer from air hunger, dyspnœa and tachypnœa. They are usually apathetic and quiet, and disinclined to feed. Such animals are nevertheless capable of considerable physical exertion. Still further from the explosion, in a zone of lower pressure, animals are unaffected externally by the explosion, although internal examination reveals changes in thoracic, and occasionally abdominal, organs. Animals exposed beyond this zone of pressure exhibit neither external nor internal effects of the explosion.[6]

The most striking feature found on post-mortem examination of animals that have died from blast is the condition of the lungs. There are numerous pulmonary hæmorrhages, varying in degree according to the severity of the blast, and showing out particularly along the line of the ribs. The hæmorrhages are due to tearing of the alveolar walls and consequent rupture of the alveolar capillaries. They are the source of the blood which is often found in the bronchi, trachea, nose and mouth.

An attempt has been made to explain the factors responsible for the pulmonary hæmorrhages.[5] The most probable cause is the sudden elevation of pressure, which compresses the chest and puts on the poorly supported pulmonary capillaries a strain greater than they can withstand. The damage done in this way may be accentuated by the suction wave, which would at first pull the chest wall outward and stretch the lung tissue.

The abdominal organs are much less sensitive to blast than are the thoracic. The large intestine is the viscus most frequently affected, the lesions ranging from small subserous hæmorrhages to perforation. The small intestine and stomach may sometimes be affected in a similar way; the liver may be bruised or torn, and occasionally there are hæmorrhages in other abdominal organs.

Damage to the nervous system is rather uncommon, but hæmorrhages do occur from both the meningeal vessels and the vessels supplying the nervous tissue. A careful search for damage to the nervous tissue has been made in the hope of finding an explanation of sudden death from blast, but animals often die from the explosion without having a demonstrable lesion in the central nervous system.

Rupture of the ear-drums occurs in animals exposed to high pressures.

Blast Injuries in Man.

There have been two important difficulties in obtaining information about blast injuries in man: (a) the condition is not easy to diagnose clinically and can be misdiagnosed even at post-mortem examination; and (b) "pure" blast injuries are rare, since the man who is exposed to blast is likely to be thrown against some solid object, or hit by a missile, or crushed by débris.

The following conditions, all of which may result from air raids, have to be diagnosed from blast effects:[2]

1. *Carbon Monoxide Poisoning.*—This may be the cause of death of an air-raid victum who does not show any signs of external injury. Asphyxia from carbon monoxide causes pulmonary congestion, and usually some pulmonary hæmorrhages occur, but spectroscopic examination of the blood establishes the diagnosis. Asphyxia from compression may also occur without gross wounding, and cause difficulty in the diagnosis of blast.

2. *Lacerations or Contusions of the Lung.*—A blow on the chest may not produce any external injury and yet cause pulmonary contusions or lacerations. The areas involved are localized, however, and their position is determined by the direction of the force causing the injury.

3. *Fat Embolism.*—It is now known that fat embolism can be caused by trauma which does not result in fracture of a bone. The lungs in such cases show patches of hæmorrhage.

4. *Shock added to Chronic Ill Health.*—People who have poor health as a result of diseases such as chronic heart disorders may be easily killed by the shock or exertion associated with an air raid.

In view of the rarity of cases of "pure" blast, and in view of the difficulty in diagnosing them, there is not yet a unanimous opinion about either the clinical findings or the pathological appearances.

Clinical Features.

The circumstances of the injury form an important part of the history, because one must try to establish whether the patient was subjected to severe blast. Inquiries are made to find out the distance of the victim from the bomb crater at the time of the accident, the degree of protection and the fate of other people who had been with the patient. Zuckerman has stated that the largest bombs used up till April, 1941, would probably never cause serious blast injuries at a distance of more than 150 feet in the open; and, further, that the wall of a room gives useful protection from blast effects.[3] The finding of ruptured ear-drums is confirmatory evidence that the blast was considerable.

Severe shock, dyspnœa, cyanosis, cough with blood-stained sputum and pain in the chest may be present in serious cases of blast injury. More usually, however, the findings are much less definite: the patient has perhaps come under observation after an air raid and does not appear well enough to resume work; or the pulse rate is persistently increased, and breathlessness occurs after slight effort; or the patient goes through a period of several hours during which he is free from symptoms and then becomes dyspnœic and coughs up some blood-stained, frothy sputum.

Moist sounds may be heard on auscultation over areas where hæmorrhages are superficial, but otherwise there are no constant physical findings; the percussion note is normal. The key to the diagnosis is the taking of a skiagram. The areas of hæmorrhage give shadows that have a fluffy indistinct margin and are fairly evenly scattered throughout both lungs.

If death occurs it frequently takes place within twelve hours. The patient who lives longer than this usually survives the blast injury, and the hæmorrhages are absorbed within a fortnight.

The above description of the clinical features applies to the so-called "pulmonary blast" which is the most common manifestation of blast injury. Occasionally organs other than the lungs are affected. This is the rule rather than the exception in immersion blast injuries, which are described later in this article.

Pathology.

There is no essential difference between the human pulmonary hæmorrhages in blast cases and the lesions that have been described in experimental animals, except that the hæmorrhages in human cases are not specially prominent beneath the ribs. Generalized capillary dilatation, however, is found in human cases, and actual tearing of the alveolar walls is rare. These two features are not found in experimental animals.

Treatment.

The essentials of treatment are rest, warmth and the administration of oxygen and morphine. Patients with blast injuries do not stand transport well. If they require an operation, either local or intravenous methods of anæsthesia should generally be used, but this is a rule which may sometimes have to be broken, just as in other branches of surgery an inhalation anæsthetic is sometimes given to a "poor risk". On account of the pulmonary vasodilatation, intravenous administration of fluids is usually contraindicated.

Special Features of Immersion Blast.

The problem of blast effects in water is simpler than that of blast on land, because only the wave of positive pressure has to be considered.[4] There are, therefore, no shattering effects from water blast. The fact that pulmonary lesions in the two instances are of the same type is a strong argument that Zuckerman was right in attributing the lesions from bomb blast to the wave of increased pressure.

Immersion blast affects the abdomen mainly, the chief lesions being rupture of the intestinal vessels and perforation of the bowel. Occasionally the eye is injured, as shown by simple chemosis, or by hæmorrhage into the anterior or posterior chamber. The lungs are often affected, but usually only in the bases. There is some evidence that a man who is swimming on his back when the depth charge explodes is much less liable to be injured than one swimming prone. It is possible that a life jacket made of buoyant material will prove of great protective value.

Conservative treatment of the abdominal injury has been successful in the majority of cases, though, of course, the possibility of a bowel perforation should be remembered. Treatment of the pulmonary injury is the same as that given above. The importance of refraining from exertion after a blast injury is shown by an incident reported by Williams.[6] Seven men on board a ship off Norway were near the magazine hatch when the ship blew up. All managed to reach the shore, but one was so ill that the other six carried him to a school where they

proposed to sleep for the night. The man who had been carried to shelter awoke next morning to find his six comrades dead.

CRUSH INJURIES.

Crush injuries, like blast injuries, have come into prominence as a result of the air raids of modern warfare. It is true that the Germans described the crush syndrome during the Great War, and that some at least of its features must have been noted by doctors after such accidents as those in collieries; but modern knowledge of the syndrome can be said to date from 1941, when Bywaters and Beall published an account of four patients who had crush injuries which led to impairment of renal function.[5]

The characteristic history they obtained was that the patient had been trapped for several hours with some object pressing on a limb. Immediately after rescue he appeared to be in good condition unless he had some associated injury. The only ill effects from the compression in the early stages were that the affected limb was swollen, and showed some wheals and local anæsthesia; the wheals were due to the return of the circulation to vessels that had been damaged by pressure, and the anæsthesia was due to direct pressure on nerve fibres or trunks. Before long the patient passed into a condition of traumatic shock: the hæmoglobin value became raised; pallor, coldness and sweating were noted; and, soon after, the blood pressure fell. Treatment of the shock by transfusion was usually successful in restoring the blood pressure.

Meanwhile the limb continued to swell; it became so tense that it could not be indented, and the circulation was impaired by the compression. Amputation sometimes became necessary, but such treatment had little influence on the rate of development of the next phase—renal failure. The urinary output, previously small on account of the shock, became smaller still, and the urine contained albumin and dark granular casts. The patient was alternately drowsy and anxiously aware of his illness. Slight generalized œdema, thirst and incessant vomiting developed and the blood pressure was often raised. Abdominal or lumbar pain sometimes occurred on about the fourth day, possibly on account of stretching of the renal capsule. The blood urea and blood potassium became progressively higher, though both were raised somewhat during the stage of shock; the blood phosphate also rose, and the carbon dioxide combining power fell. Tubular dysfunction was shown by a failure to concentrate urea or to reabsorb chlorides. Death from renal failure occurred suddenly at about the end of the first week.

The three outstanding features of the syndrome, therefore, are the comparatively good condition of the patient when rescued, the onset of shock, which, however, responds to transfusion, and the development of renal failure. Death does not invariably occur; the end of the first week is the critical period; in those who recover, a sudden diuresis occurs about that time. The patients who survive amount to about one-third of those who are sufficiently ill to be treated in hospital.

Pathology of Crush Injuries.

The outstanding lesions seen at post-mortem examination of fatal cases are muscle necrosis and degeneration of the renal tubules.[7]

The muscle necrosis may take one or more of three forms: (a) The muscle may be injured by direct pressure and be blanched and friable, like fish flesh. The injured muscle in this case lies directly beneath the injured skin. (b) The lesion may be due to rise of pressure within a fascial sheath, in which case the whole of the muscle becomes white and necrotic. (c) The injury to the muscle may be produced by spasm of the arteries, which, in turn, is due to periarterial hæmorrhage or direct arterial damage; this type may therefore be produced without a long period of compression. The muscle is of normal colour and appearance on macroscopic examination, but microscopically it shows patchy necrosis of isolated fibres.

The kidneys are enlarged except in patients who die early. The glomeruli are normal, but the tubules show degeneration. The lumina of the tubules are blocked by orange-coloured or brown granular casts; these occur only in the distal part of the tubule in which the urine changes to an acid reaction. In some cases, after three or four days areas of focal necrosis appear in the boundary zone: large hyaline casts are extruded into the interstitial tissue and these are surrounded by an œdematous area in which cellular proliferation occurs.

The renal lesions are not specific to crush injuries, but occur following intravascular hæmolysis from various causes.

Pathogenesis.

The method by which the lesions are produced is of the greatest interest, because it has determined the type of therapy used. There are at least three possible ætiological factors concerned in the causation of the syndrome:

1. The compression syndrome might be due to a loss of blood constituents from the circulation into the tissues of the injured limb.[11] The capillaries temporarily lose their impermeability, owing to the prolonged compression, and permit plasma proteins to pass through their walls. The œdema of the affected limb is often so pronounced that small abrasions of the skin weep copious amounts of plasma.

This factor comes into operation when the compressing weight is removed, and it is undoubtedly the precipitating cause of the shock which occurs at that time. It is important to remember, however, that except in neglected patients, renal failure, and not shock, is the cause of death in compression injuries. The renal failure has never been adequately explained by the loss of fluid at the site of injury and the importance of this loss is therefore lessened.

2. One of the striking features of muscles injured by long compression is their pallor. This is due to the loss of myohæmoglobin, the molecule of which is one-quarter the size of the molecule of circulating hæmoglobin. It has been demonstrated that the pigment precipitated in the renal tubules is acid myohæmoglobin. This pigment is not found in the blood stream because it has a low renal threshold and passes readily through the glomerular membrane.

These findings suggest that the kidney damage in patients with crush injuries is similar to that caused by a hæmolytic transfusion reaction. There is no convincing evidence as yet that the damage is due to a mechanical blockage of the tubules, but this can be said also of the "transfusion kidney". The work on the rôle of muscle hæmoglobin has at least shown a relation between the new syndrome of crush injury and the familiar picture of a hæmolytic transfusion reaction.

3. It has been suggested that some unidentified toxic substance might be liberated from necrotic muscle and cause the kidney damage.

In an experimental study on dogs[9] the findings were that local fluid loss and absorption of toxins were each important factors. The former was the more important when the compression was of moderate duration; the latter increased in significance when the compression was prolonged.

It is worth recording that some critics were disinclined at first to regard the compression syndrome as a new entity, and thought it to be due to multiplicity of transfusions of blood and serum. This is no longer tenable. In the case of some of the original patients reported, serum and blood transfusions had been given, but the syndrome has since occurred in patients who were given no other therapy than quiet rest. Moreover, of the many shocked patients whom Bywaters has treated by massive transfusions, only those with compression injuries ever showed the compression syndrome.[7]

Differential Diagnosis.

There are several conditions found amongst air-raid casualties which may be confused with crush injury. They can be discussed according as to whether they produce the important signs of oliguria, hæmaturia or swelling of an injured limb.

Oliguria is most frequently due to traumatic shock alone and therefore disappears if the blood pressure is restored to a normal value. It may also be caused by dehydration, sulphonamide therapy or incompatible transfusion. Retention of urine, as distinct from oliguria, occurs in the rare condition of cortical necrosis following trauma to the liver, but the retention can be diagnosed by catheterization. Dehydration can be detected if a fluid balance chart is kept. Precipitation of sulphonamide crystals is suspected if oliguria, hæmaturia and lumbar pain develop in anyone taking the drug; the patient is investigated by cystoscopy and ureteric catheterization whenever the oliguria persists for more than twenty-four hours. An incompatible transfusion is diagnosed by noting the characteristic reaction during the transfusion, by repeating the grouping and cross-matching tests, and by examining the patient's blood for bilirubin and free hæmoglobin.

Hæmaturia may occur from injury to the kidney, and hæmoglobinuria may result, in susceptible people, from exposure to the cold. The former can be recognized by examination of the urine with a microscope; the latter is distinguished by the characteristic spectroscopic appearance of the circulating free hæmoglobin.

Swelling of the limb may be due to a hæmatoma. In this case the skin wheals are absent. There is a possibility that in the unconscious patient the effects of burns may be confused with those of a crush injury.

Incidence.

Crush injuries are much more common than those due to blast. In an urban area crush injuries may be present in 5% of air-raid casualties.[7]

Prognosis.

There is no simple inverse proportional relation between the duration of the crushing and the prospect of recovery; sometimes, indeed, brief trauma produces extensive damage.

A man of 24 developed renal failure after having been pinned for five minutes beneath an overturned Bren gun carrier. . . . The same thing happened to a man who was extricated almost immediately after a road accident. . . . Of the cases published by the Medical Research Council subcommittee . . . the average duration of burial of the five patients who recovered was nearly twice as long as that of the five who died.[10]

Of course, the degree of compression has to be taken into account in considering the time factor.[7] For any given compressing force the prognosis appears to be best when the patient is buried for either a short or a very long period. Periods of burial lasting longer than twelve hours seem to be less serious than any but the very short periods. This may be because the patients who are "poorer risks" succumb during the twelve hours, or again, it may be because the injury is so severe that the circulation to the affected part is not reestablished and a form of natural amputation takes place.

Patients with crushing injury of the arm have a better prognosis than those whose leg has been involved. The blood urea is a useful pointer to the patient's progress. It is a favourable sign if the blood urea either decreases or, at any rate, rises only slowly. The degree of hæmoconcentration and the urinary output are also of value in assessing prognosis. Occasionally a patient who has been given a poor prognosis by all available criteria recovers unexpectedly without treatment, and claims of successful therapy, therefore, have to be closely scrutinized.

Treatment.

It must be remembered that prophylactic treatment for the kidney failure is the most important thing to consider. Although this failure does not become obvious before the third day, all theories agree that the kidneys have probably started to suffer damage by the time the patient comes under treatment.

The measures taken against renal failure are as follows:

1. Ample alkaline fluid is given with the idea of preventing the precipitation of acid myohæmoglobin. The doses of alkali required are large, since the reaction of the urine must be quickly made alkaline and retained at a high pH. If possible it is best to start by giving intravenously ten cubic centimetres of twice molar sodium lactate solution and ten cubic centimetres of a saturated solution of sodium bicarbonate. The urine becomes alkaline in fifteen minutes and remains so for at least one and a half hours. An alkaline mixture may then be given by mouth and its effect judged by testing the reaction of the urine. With these large doses of alkali maintained for over a week there is some danger of inducing tetany, and calcium gluconate should therefore be kept on hand.

2. Those who believe that toxins are absorbed from the damaged area sometimes advocate that a tourniquet or tight bandage should be applied as a first-aid measure to prevent this absorption.

Shock is the second most important manifestation to be treated. If fluid is given intravenously, a large proportion of it drains into the injured limb, which is toneless and flaccid and has an enormous fluid capacity. It is therefore recommended that the affected limb should be bandaged during first-aid treatment with an elastic web bandage.[11] This prevents further œdema and assists in the reabsorption of any fluid that has already escaped.

In addition, the usual treatment for traumatic shock is given. The patient has had a trying ordeal and requires morphine to ensure mental and physical rest. Blood or a blood derivative is necessary to restore the circulatory volume.

In some instances amputation is indicated. When this operation is necessary it should be done as early as possible. If delayed beyond the first day the removal of the limb gives little protection from renal failure, and should be undertaken only if the limb is non-viable.

Usually the limb will be retained. It should be elevated and packed round with ice. The elevation favours venous and lymphatic return, and the ice delays autolysis and decreases the metabolic needs of the part. Sometimes the use of multiple incisions through the deep fascia may relieve the pressure.

Many patients with crush injuries have other conditions requiring treatment. Soft tissue injury and fractures are commonly present. Other complications are hæmorrhage, fat embolism, blast and carbon monoxide poisoning.

Even if the patient's life is saved there is some disability in the form of damage to the muscles. Fibrous tissue is formed during recovery and there is a danger of contractures developing; this should be avoided by splinting and exercise.

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